MedTrain
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Pain management
معالجة الألم

مستشفى جامعة العلوم والتكنولوجيا
University of Science & Technology Hospital
ملتزمون بالتميّز

PAIN MECHANISM AND ASSESSMENT

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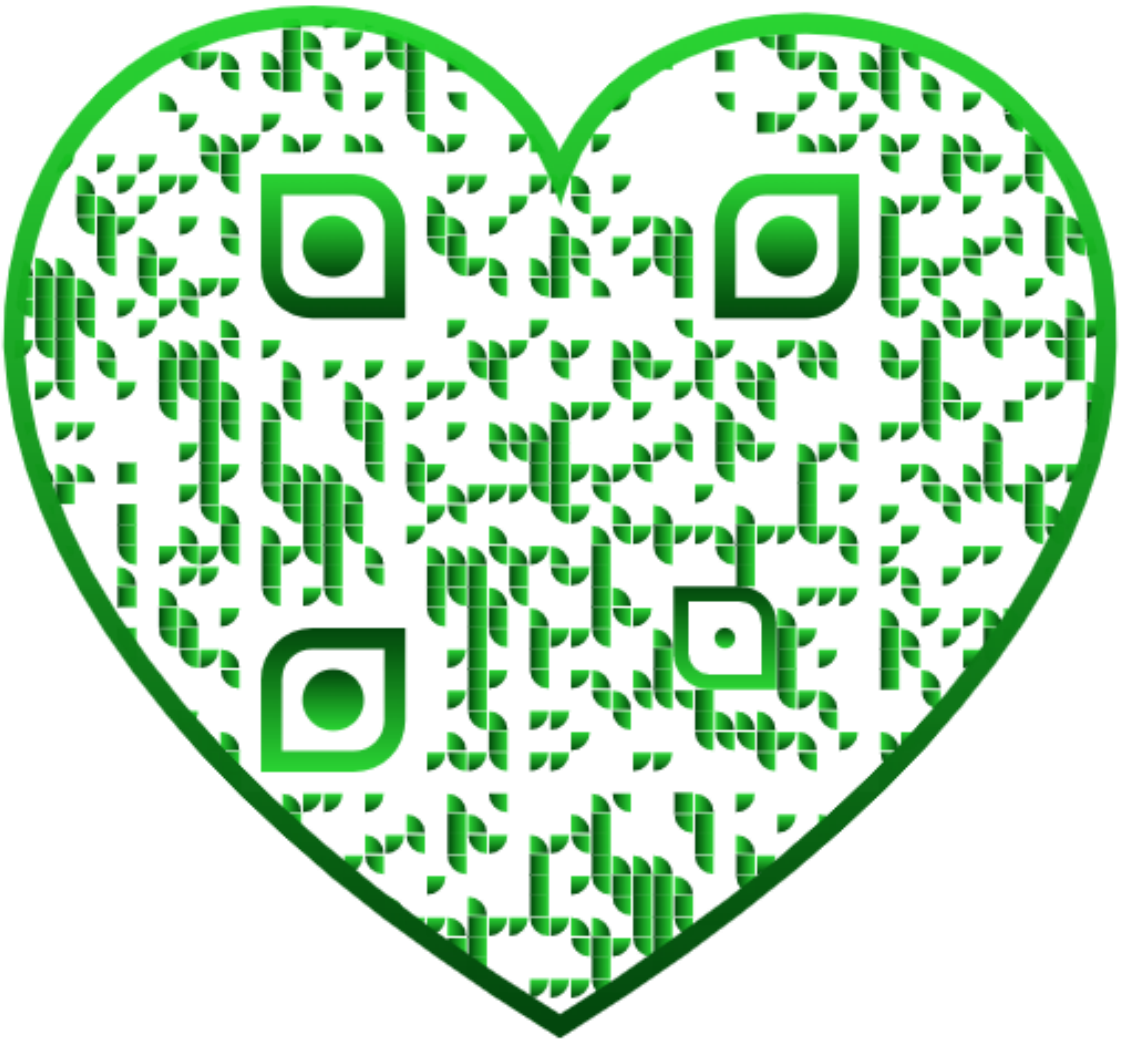
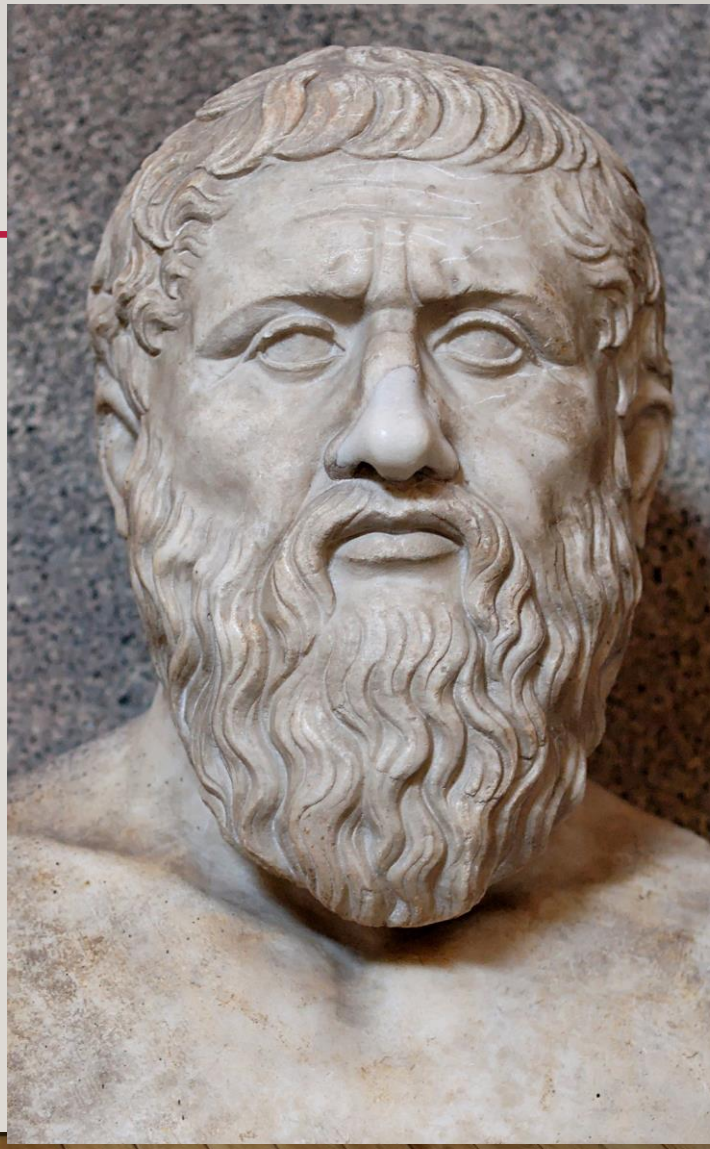


Athenian philosopher, Plato (c. 428 to 347 B.C.), who defined pain as an 'emotion' that occurs when the stimulus is intense and lasting . Plato called it the “Intensity Theory.. Centuries later, we are aware that especially chronic pain represents a dynamic experience, profoundly changeable in a spatial-temporal man

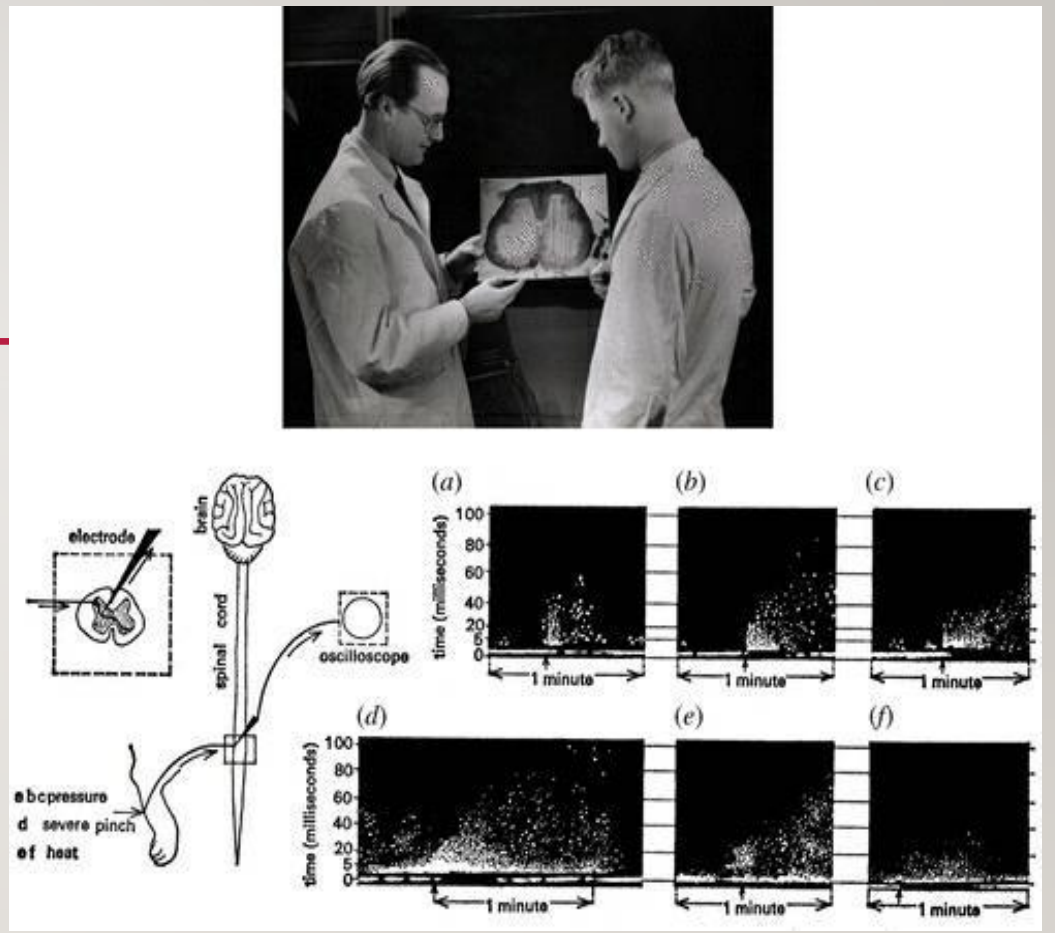


The oldest explanation for why pain manifested in specific populations was due to **bad air, motion of planets and mainly rooted in religious beliefs**. Throughout history, religious ideologies have had a substantial influence on people's thoughts and actions. As a result, the majority of people believed that pain was the consequence of **committing immoral acts**. There was also a belief that the suffering they endured was the individual's way to repent for these sins. Although this belief remained popular up until the nineteenth century

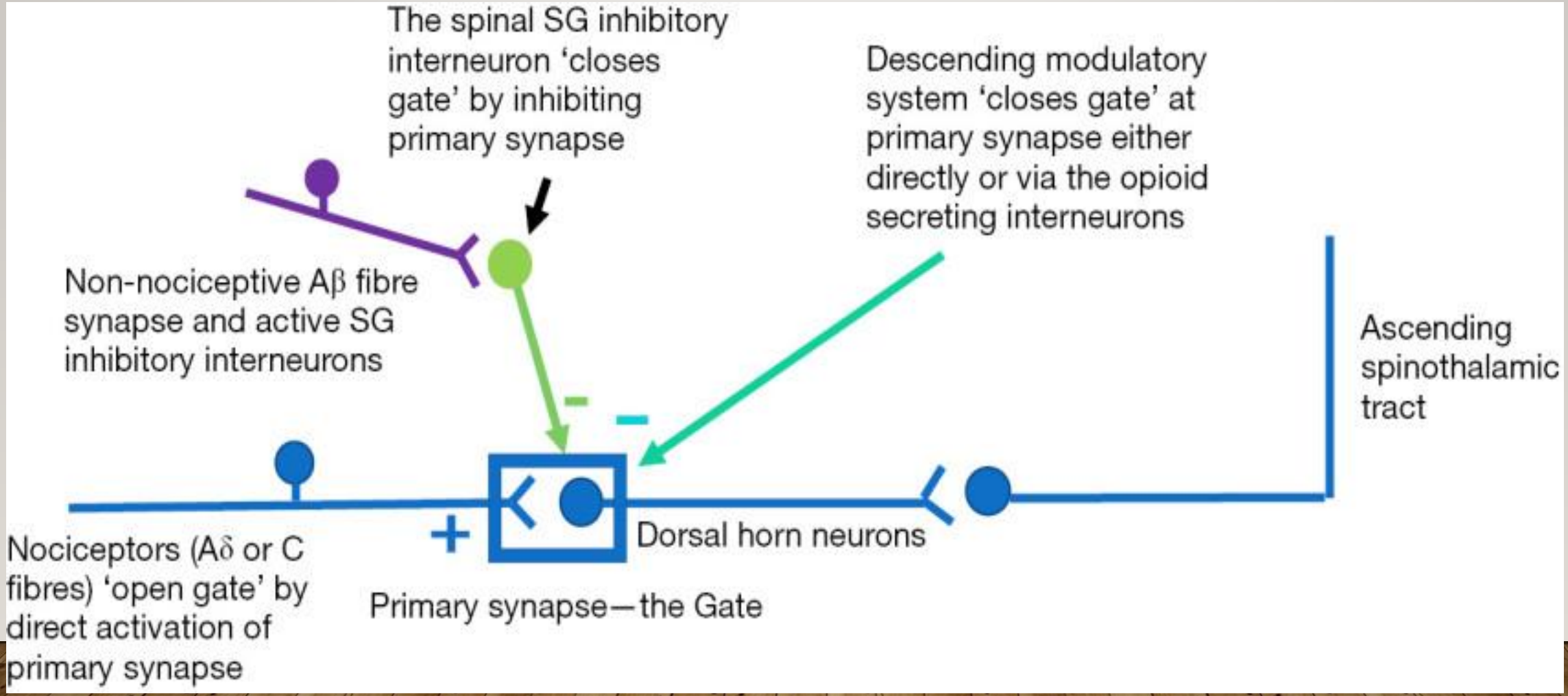
- In the early 1600s, French philosopher René Descartes (1596-1650) proposed the "**Cartesian Dualistic Theory**" which suggests that pain could either result from physical or psychological injury however were not influenced by each other. Thus, the belief was that physical and psychological factors were independent determinants of pain and did not combine to create a synergistic effect

It wasn’t until the nineteenth century, after a series of experiments, that a scientific basis for pain was established. **The “Specificity Theory**” was initially presented by Charles Bell (1774-1842) in 1811 . Bell’s theory allowed the brain to be introduced as a complex structure with an abstract role by presenting the idea that specific input pathways may be assigned to each of the different sensations.

In 1965, Patrick David Wall (1925–2001) and Ronald Melzack announced the first theory that viewed pain through a mind-body perspective. This theory became known as **the gate control theory.** Melzack and Wall's new theory partially supported both of the two previous theories of pain but also presented more knowledge to advance the understanding of pain further. The gate control theory of pain states that when a stimulus gets sent to the brain, it must first travel to three locations within the spinal cord. These include the cells within the substantia gelatinosa in the dorsal horn, the fibers in the dorsal column, and the transmission cells which are located in the dorsal horn.



GATE CONTROL THEORY


The spinal SG inhibitory interneuron 'closes gate' by inhibiting primary synapse
Descending modulatory system 'closes gate' at primary synapse either directly or via the opioid secreting interneurons
Non-nociceptive Aβ fibre synapse and active SG inhibitory interneurons
Ascending spinothalamic tract
Dorsal horn neurons
Nociceptors (Aδ or C fibres) 'open gate' by direct activation of primary synapse
Primary synapse—the Gate

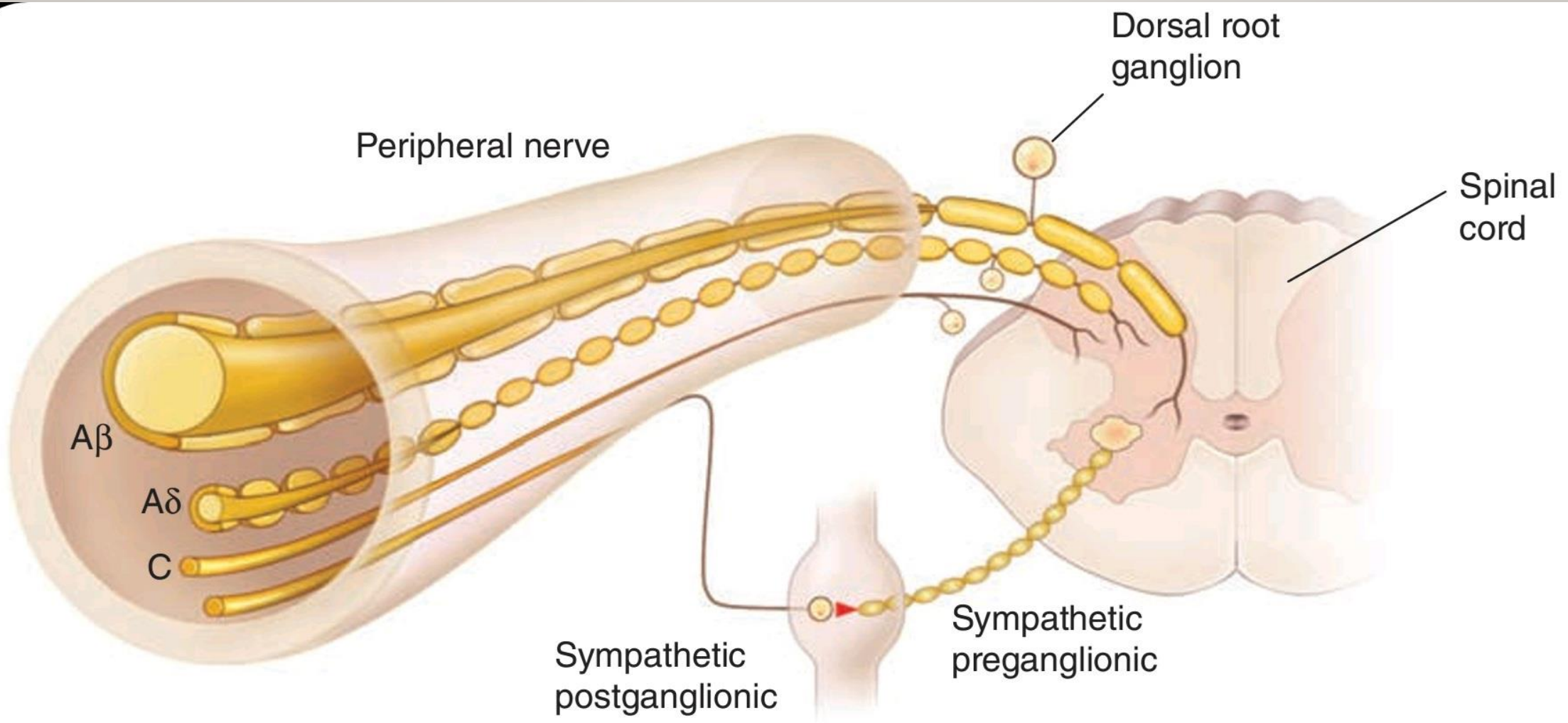
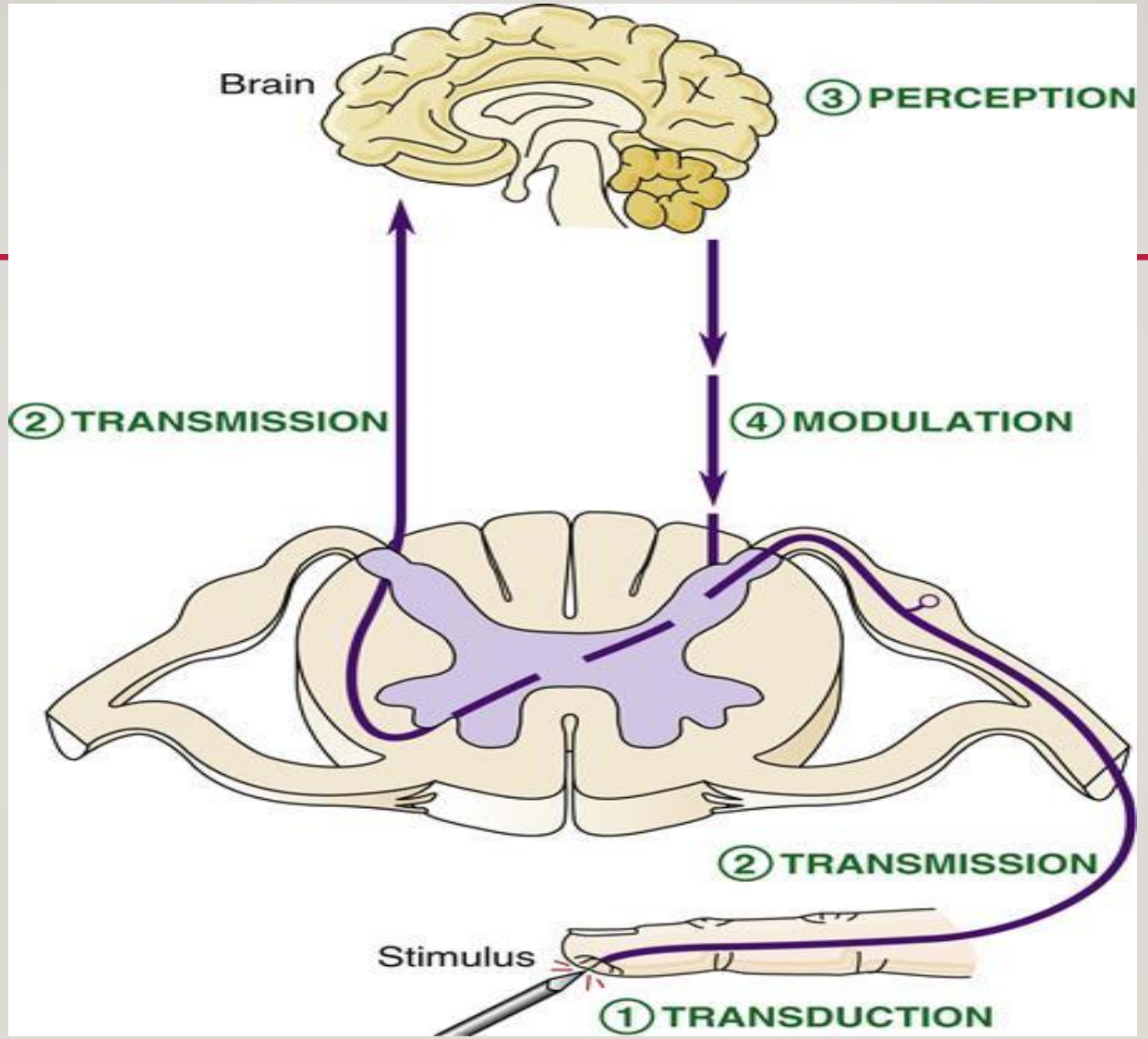


FIGURE 13-1 Components of a typical cutaneous nerve. There are two distinct functional categories of axons: primary afferents with cell bodies in the dorsal root ganglion and sympathetic postganglionic fibers with cell bodies in the sympathetic ganglion. Primary afferents include those with large-diameter myelinated (Aβ), small-diameter myelinated (Aδ), and unmyelinated (C) axons. All sympathetic postganglionic fibers are unmyelinated.

THE PHYSIOLOGICAL PROCESSES OF PAIN MECHANISM



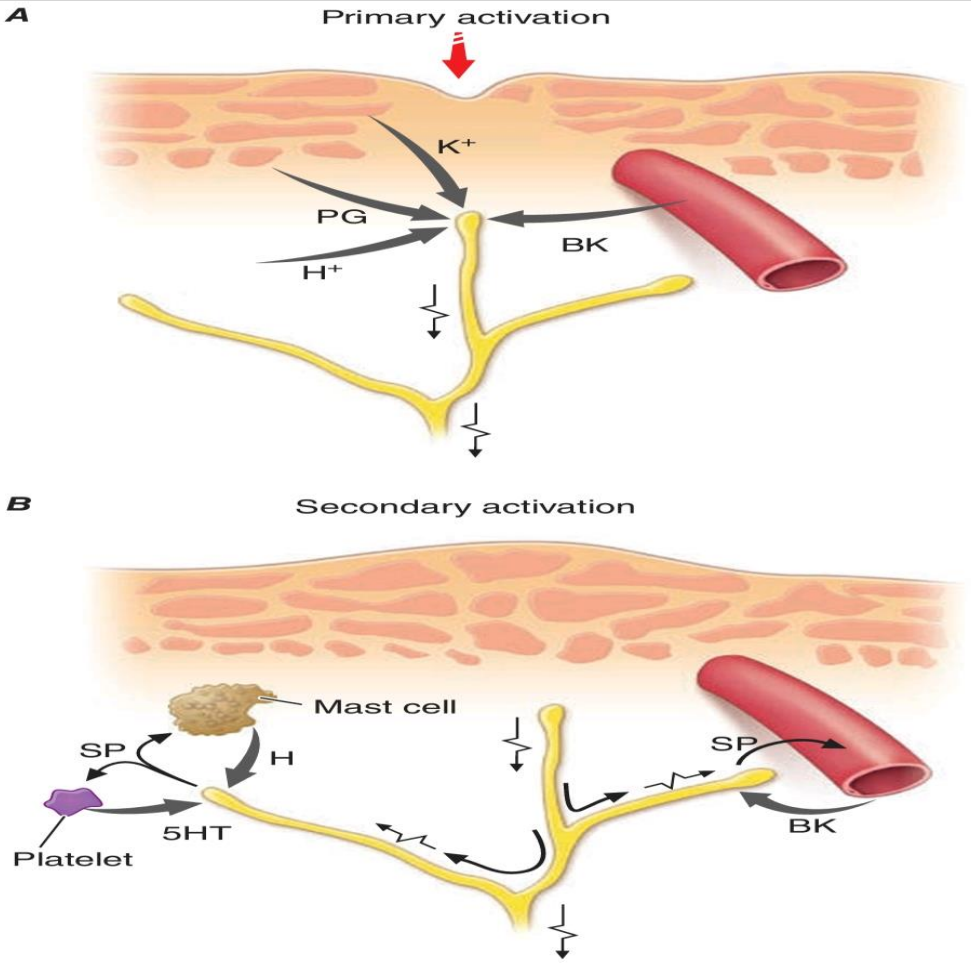


FIGURE 13-2 Events leading to activation, sensitization, and spread of sensitization of primary afferent nociceptor terminals. ***A***. Direct activation by intense pressure and consequent cell damage. Cell damage induces lower pH (H^+) and leads to release of potassium (K^+) and to synthesis of prostaglandins (PGs) and bradykinin (BK). PGs increase the sensitivity of the terminal to BK and other pain-producing substances. ***B***. Secondary activation. Impulses generated in the stimulated terminal propagate not only to the spinal cord but also into other terminal branches where they induce the release of peptides, including substance P (SP). Substance P causes vasodilation and neurogenic edema with further accumulation of BK. Substance P also causes the release of histamine (H) from mast cells and serotonin (5HT) from platelets.

34.2 Neurotransmitters and receptors involved in pain processing in the spinal cord

Neurotransmitter	Receptor(s)	Receptor type	Comments*
Amino acids			
Glutamate	AMPA	Ion channel	Excitatory; permeable to cations: can be Ca^{2+}, Na^+ or K^+, depending on subunit structure
	NMDA	Ion channel	Excitatory; blocked by Mg^{2+} in the resting state; block can be altered if membrane potential changes; permeable to Ca^{2+}, Na^+ and K^+
	Kainate	Ion channel	Post synaptic – excitatory
	Gp I	GPCR	Pre-synaptic – inhibitory through GABA release; permeable to Na^+ and K^+
	Gp II	GPCR	Activates a range of signalling pathways; long-term effects on synaptic excitability
	Gp III	GPCR	Probably inhibitory; can decrease cAMP production; pre-synaptic; decreases glutamate release
Glycine	GlyR	Ion channel	Mainly inhibitory; permeable to Cl^-; blocked by caffeine
GABA	$GABA_A$	Ion channel	Mainly inhibitory in spinal cord; permeable to Cl^-; indirectly modulated by benzodiazepines (increased ion channel opening); not specifically involved in nociception, generally depressant effect on spinal cord activity
	$GABA_B$	GPCR	Predominantly inhibitory; activated by baclofen
Neuropeptides			
Substance P	Neurokinin receptors	GPCR	Mainly excitatory; increased in inflammation, decreased in neuropathic pain
Cholecystokinin	CCKRs1–8	GPCR	Excitatory; clinical trials of antagonists in progress
Calcitonin gene-related peptide	CALCRL	GPCR	Excitatory; slows degradation of substance P; implicated in migraine
Opioids			
Dynorphin	OP1 (kappa)	GPCR	Excitatory?; may be pro-nociceptive
β-endorphin	OP3 (mu)	GPCR	Inhibitory
Nociceptin	ORL-1	GPCR	Inhibitory; also expressed by immune cells

*Excitatory = increased pain; inhibitory = reduced pain.
(AMPA = α-amino 3-hydroxy, 5-methyl, 4-isoxazole propionic acid; CALCRL = calcitonin receptor-like receptor; cAMP = cyclic adenosine monophosphate; GABA = γ-aminobutyric acid; Gp = group; GPCR = G-protein-coupled receptor; NMDA = *N*-methyl-D-aspartate; OP = opioid; ORL = opioid-like receptor)

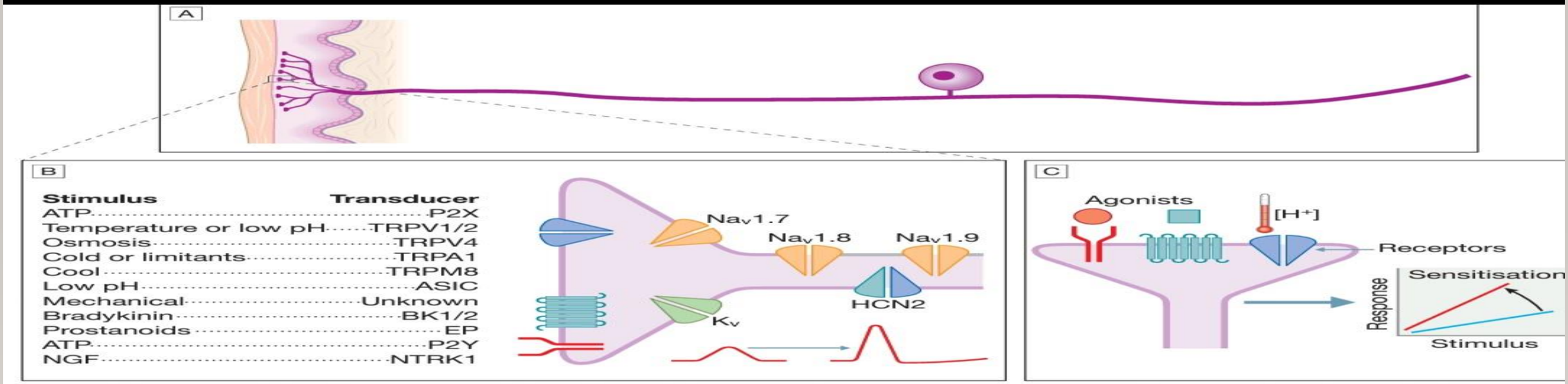


Fig. 34.3 Mechanisms of peripheral sensitisation. A Sensory nerve terminating with nociceptor in skin. B Peripheral nociceptors express various receptors and ion channels that act as mediators of pain. They include sodium channels implicated in congenital pain syndromes; the purinergic 2X (P2X) receptor for adenosine triphosphate (ATP); members of the transient receptor potential (TRP) superfamily of ion channel receptors, which detect changes i osmolality and temperature; acid-sensing ion channel (ASIC) receptors, which detect hydrogen ions; G-protein-coupled receptors, which detect bradykinin (BK), prostaglandins and ATP; and the neurotrophic tyrosine kinase 1 (NTRK1) receptor, which detects nerve growth factor (NGF). C Activation of these receptors by ligands, hydrogen ion [H^+] and high temperature (>42°C) amplifies action potentials, which increase pain signals and cause peripheral sensitisation. *Adapted from Bennett DL, Woods CG. Painful and painless channelopathies. Reprinted with permission from Elsevier (The Lancet Neurol 2014; 13:587–599).*

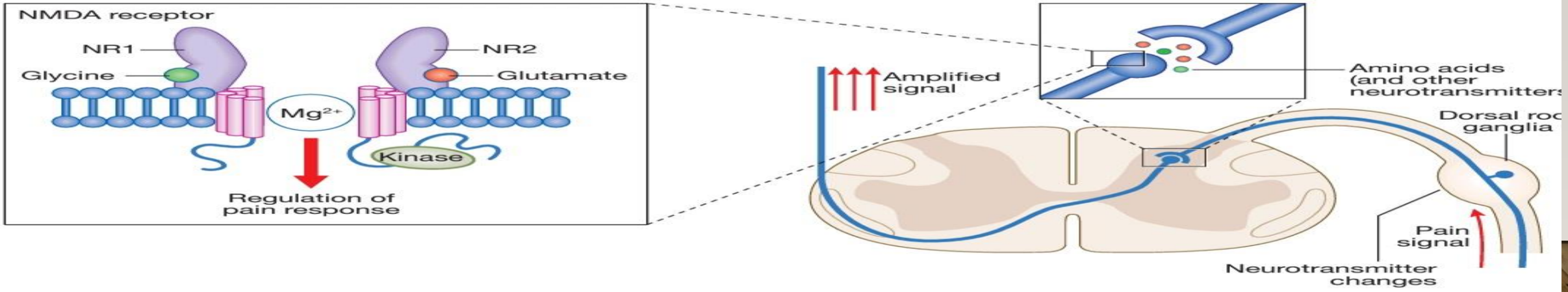
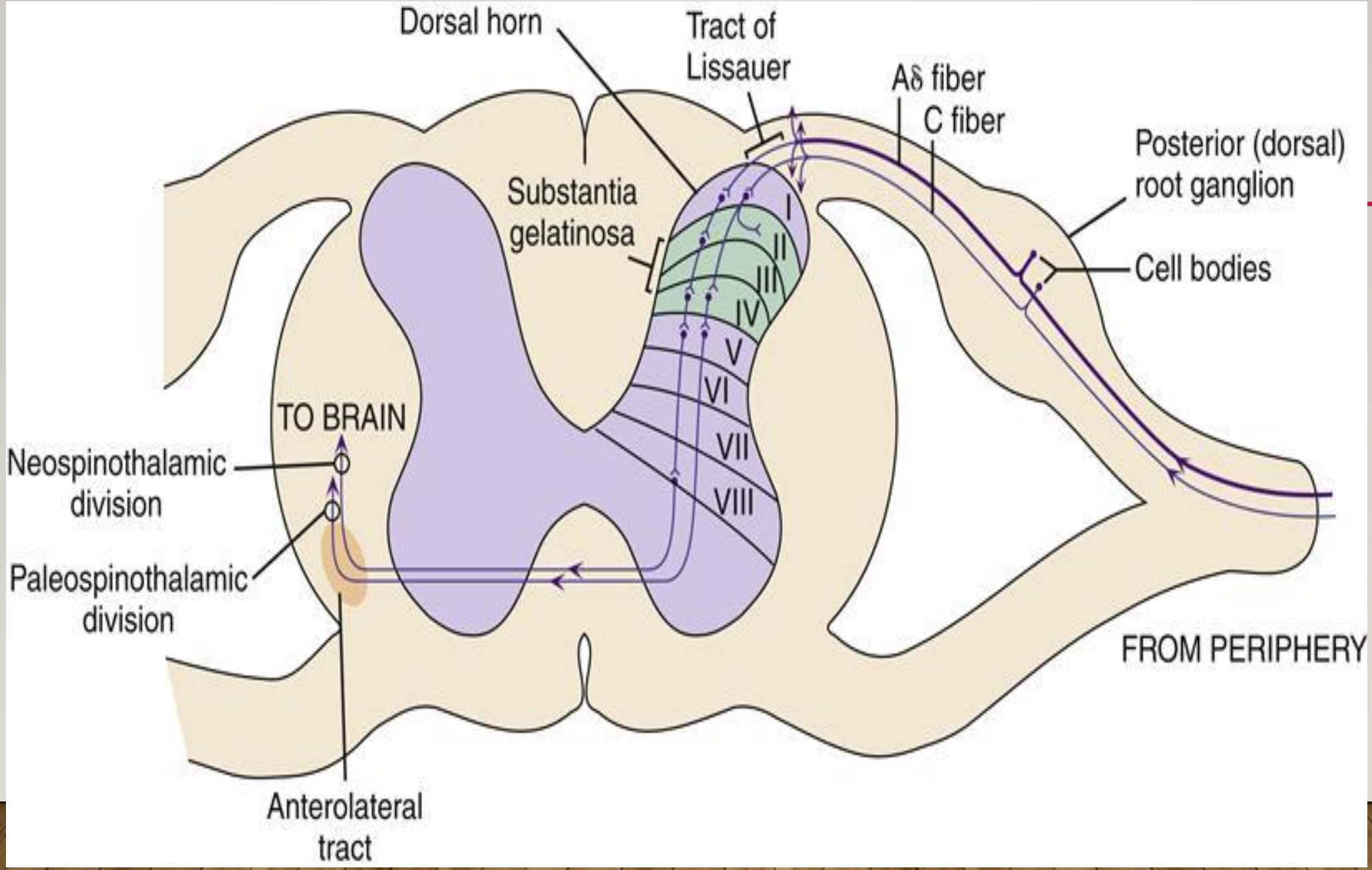
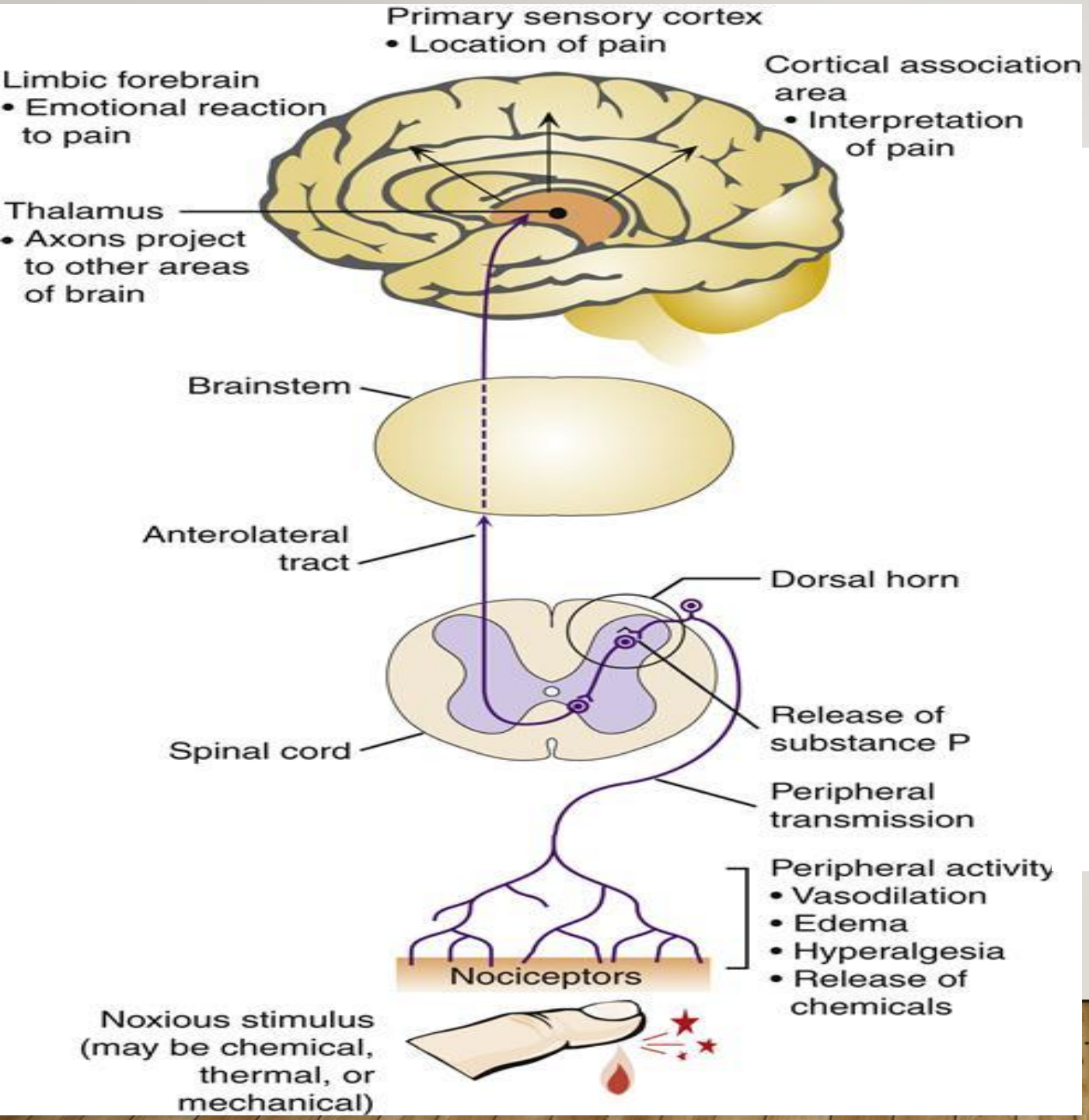
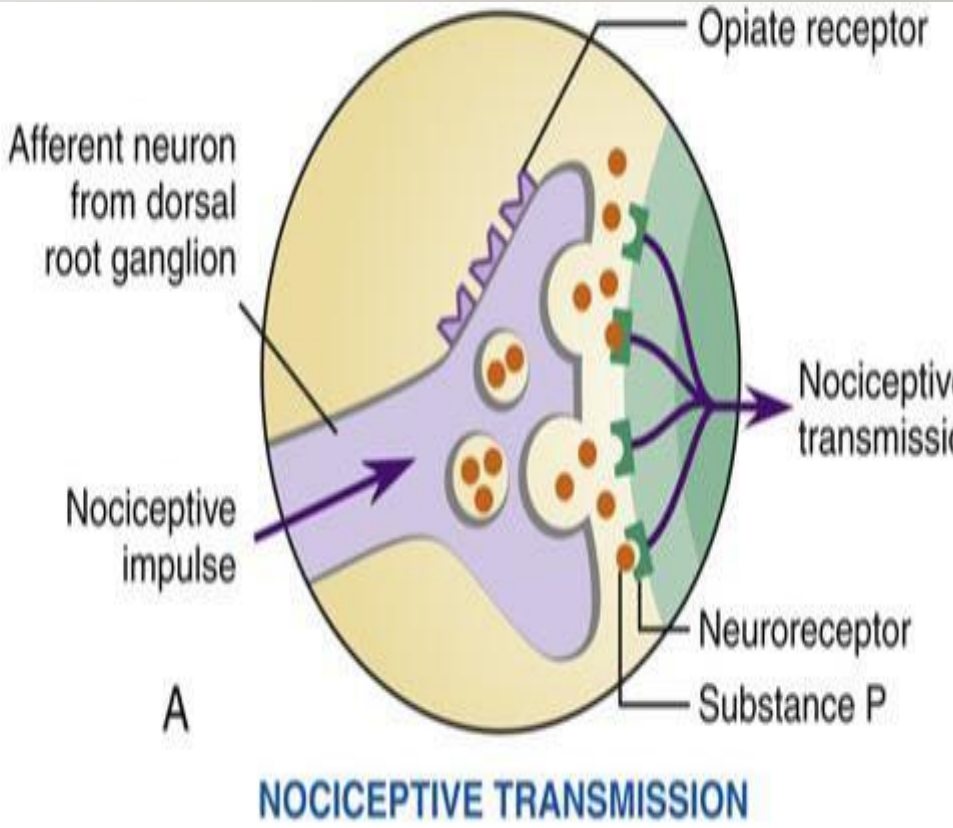


Fig. 34.4 Mechanisms of central sensitisation. Post-synaptic activation of the *N*-methyl-D-aspartate (NMDA) receptor requires the amino acids glycine and glutamate, which bind to the NR1 and NR2 subunits, respectively; these amplify pain signals at the level of the spinal cord. In contrast, magnesium ions block receptor activation.


Dorsal horn
Tract of Lissauer
Aδ fiber
C fiber
Posterior (dorsal) root ganglion
Substantia gelatinosa
I
II
III
IV
V
VI
VII
VIII
Cell bodies
TO BRAIN
Neospinothalamic division
Paleospinothalamic division
FROM PERIPHERY
Anterolateral tract





NOCICEPTIVE TRANSMISSION

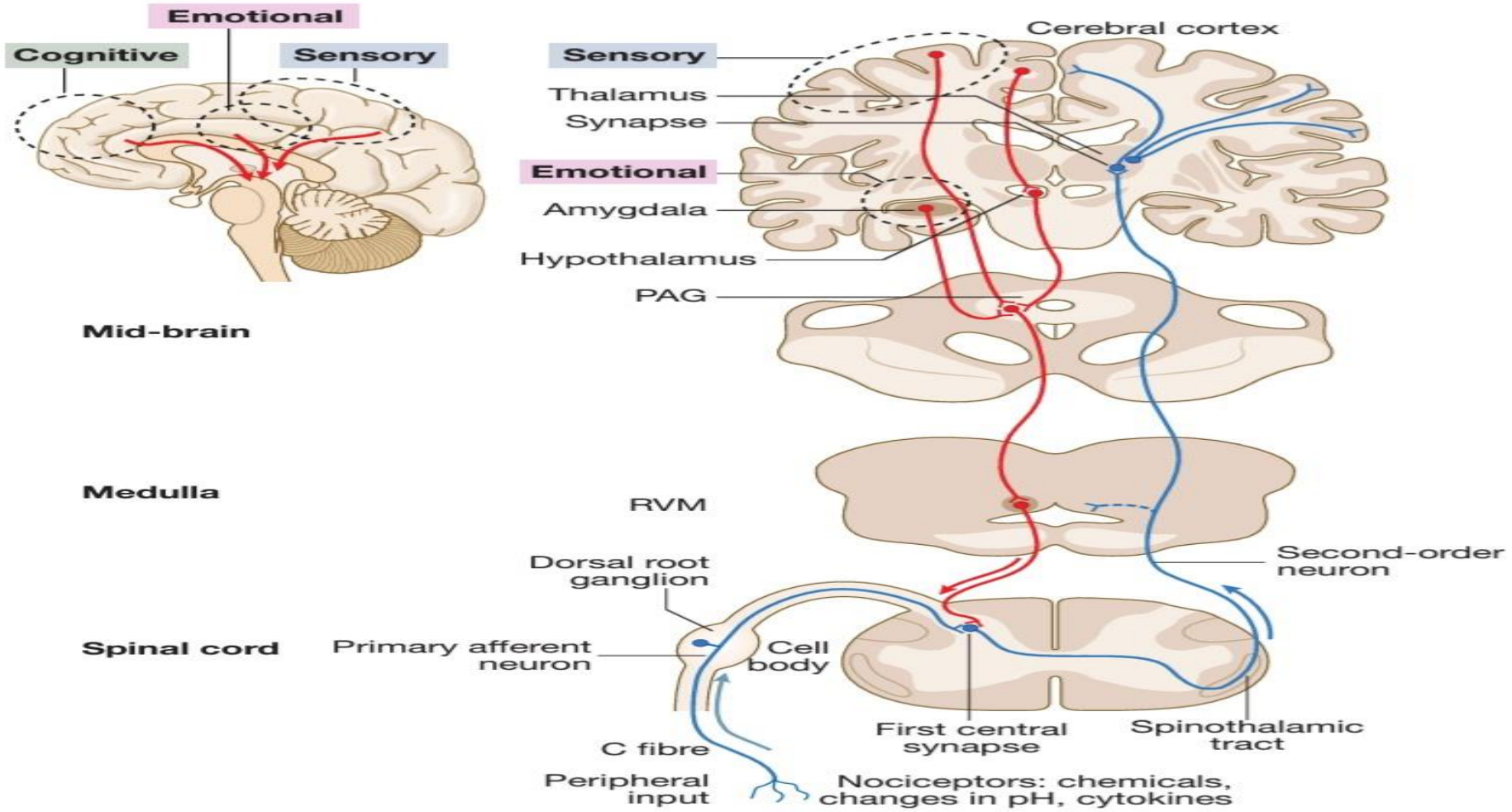
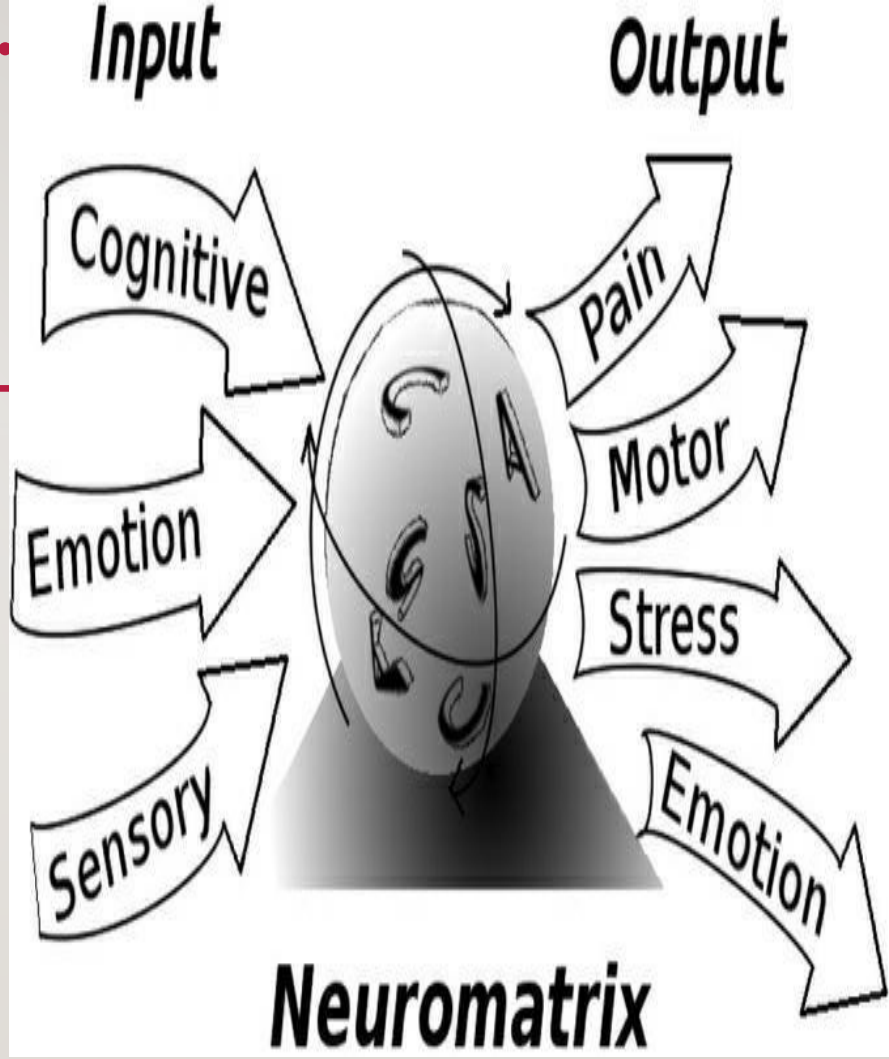
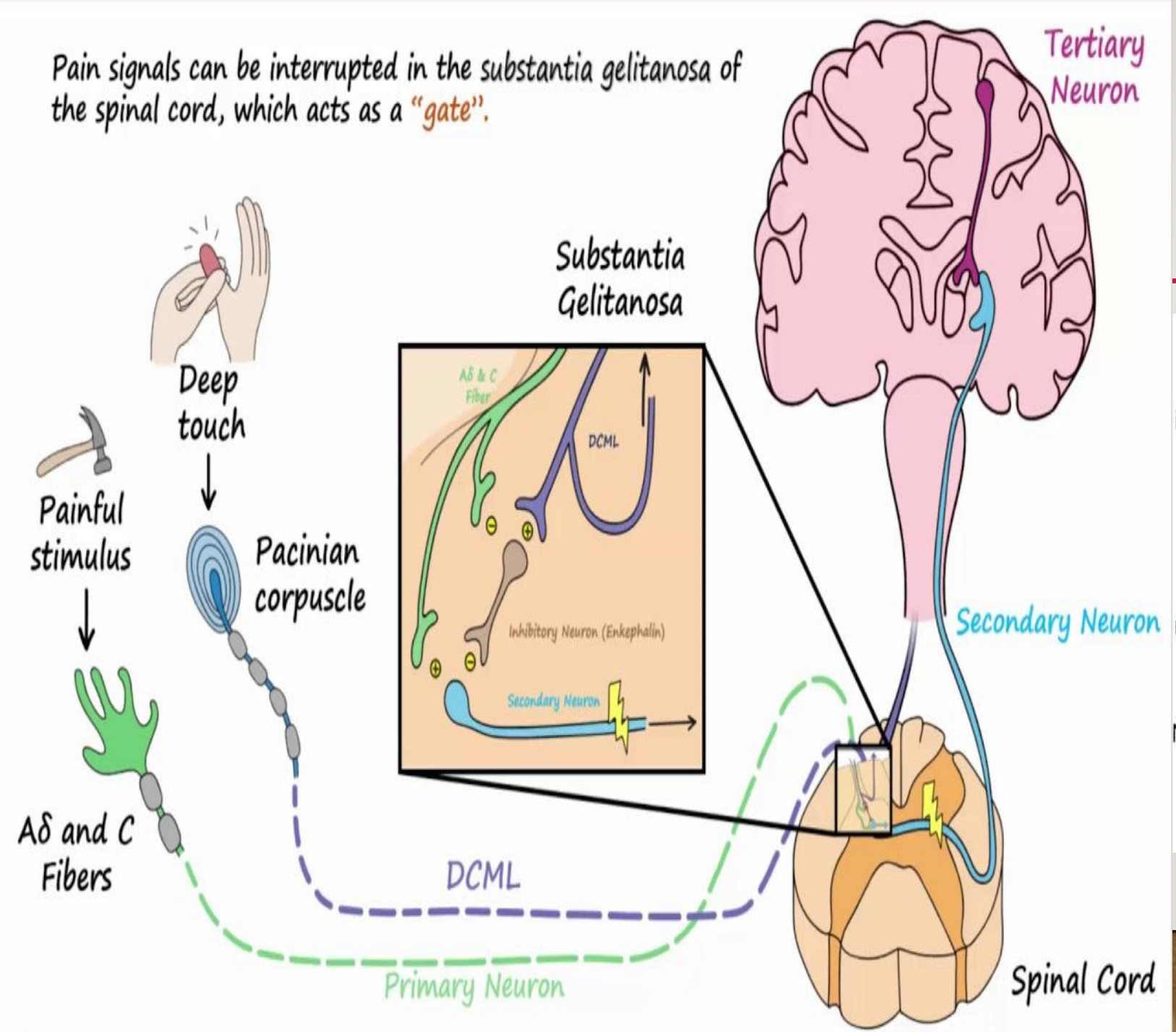


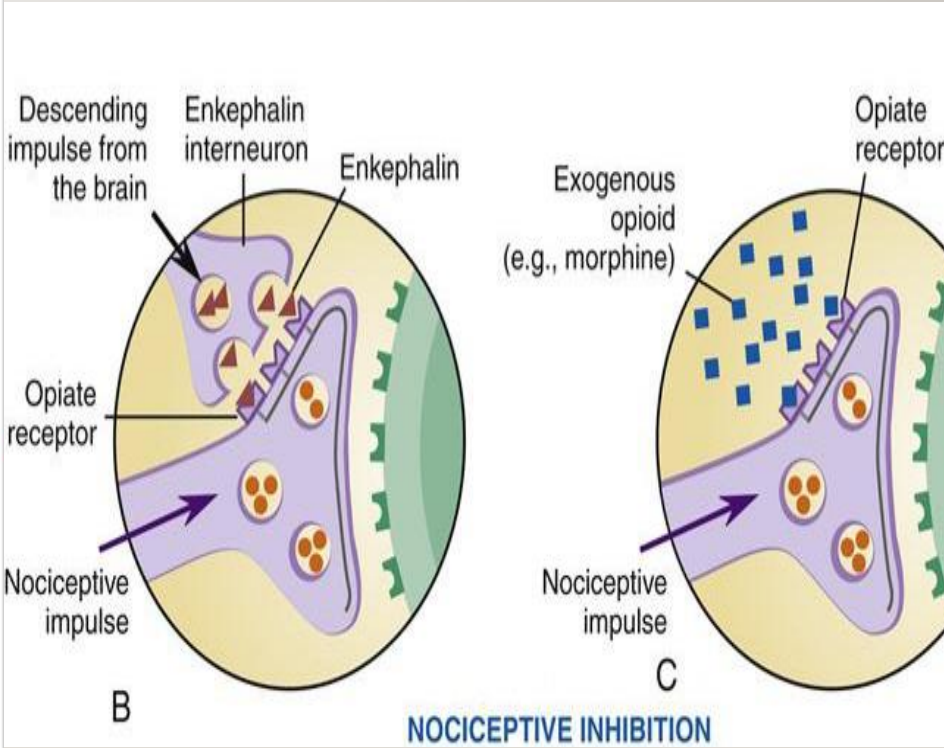
Fig. 34.2 Ascending and descending pain pathways. Ascending pathways are shown in blue and descending in red. Pain signals are detected in t periphery by nociceptors, which are activated by chemicals, changes in pH and cytokines. The signal is transmitted by the primary afferent neuron to th spinal cord, where there is a synapse with a second-order neuron, which transmits the signal onwards to the thalamus. Thereafter, the pain signal is transmitted to the cerebral cortex. The intensity of pain signals is subject to extensive modulation at several levels within the nervous system. Cognitive influences derived from the frontal lobe, coupled with sensory influences from cortex and emotional influences from the amygdyla, affect pain perceptio the mid-brain around the periaqueductal gray matter (PAG) and the rostroventrolateral medulla (RVM) in the medulla. These structures form part of the descending modulatory systems, which, under normal circumstances, inhibit pain perception. In some chronic pain states, however, dysfunction of the descending pathways can occur, increasing pain.

30 years after the pain gateway theory, Ronald Melzack's exposure to amputees suffering from phantom limb pain prompted his inquiry that led to the development of a "**Neuromatrix Model**" . This model suggests that CNS is the origin responsible for creating painful sensations rather than the periphery . The neuromatrix hypothesis states that pain is a product of different patterns of signals from these different areas in the CNS; the patterns of signals were referred to as the "neuro signature." Peripheral sensory input, including nonphysical factors, can influence the neuro signature, but cannot create its own signature . This model also acknowledges the notion that pain can be affected by cognitive and emotional factor



Pain signals can be interrupted in the substantia gelitanosa of the spinal cord, which acts as a "gate".





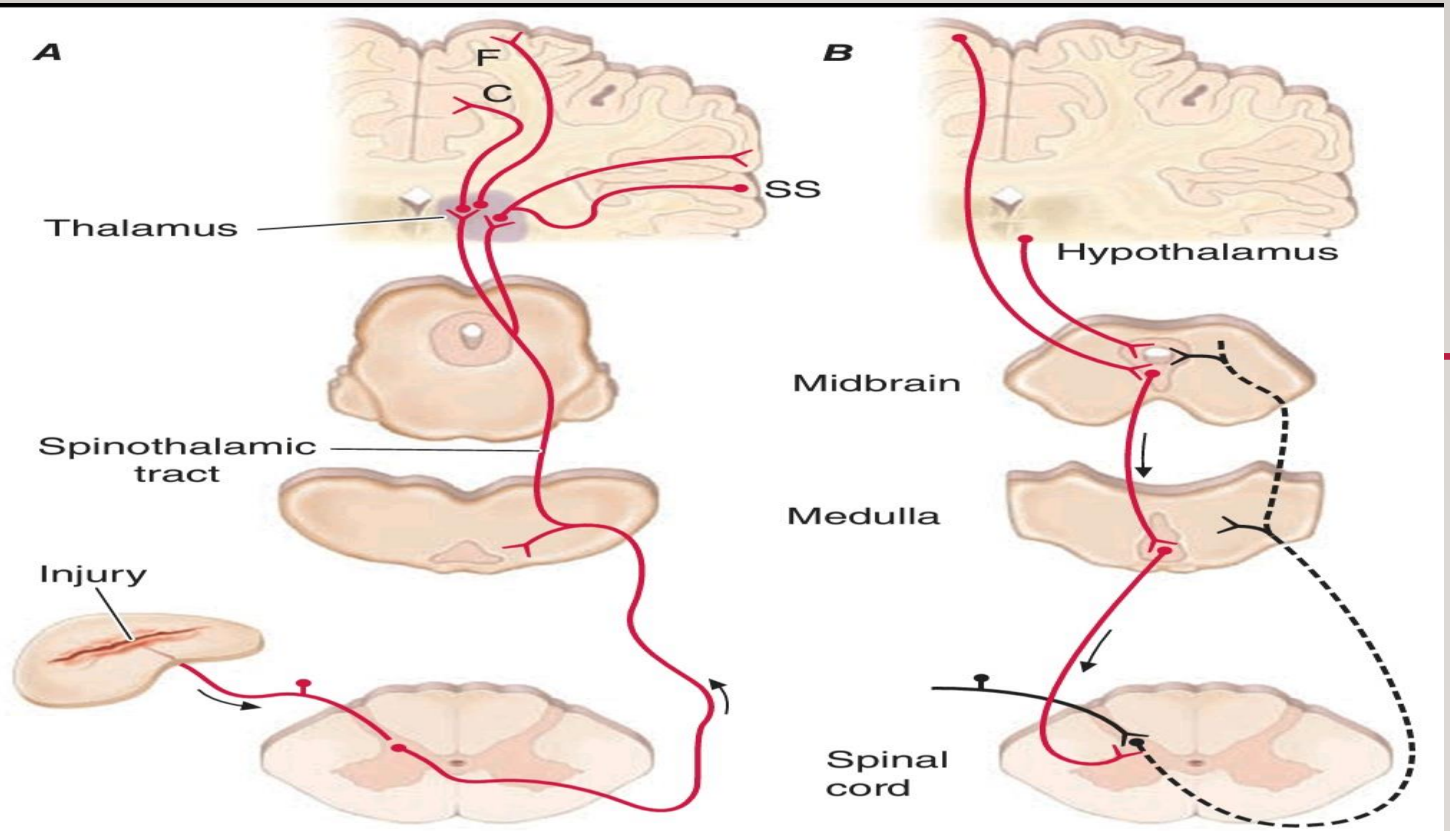
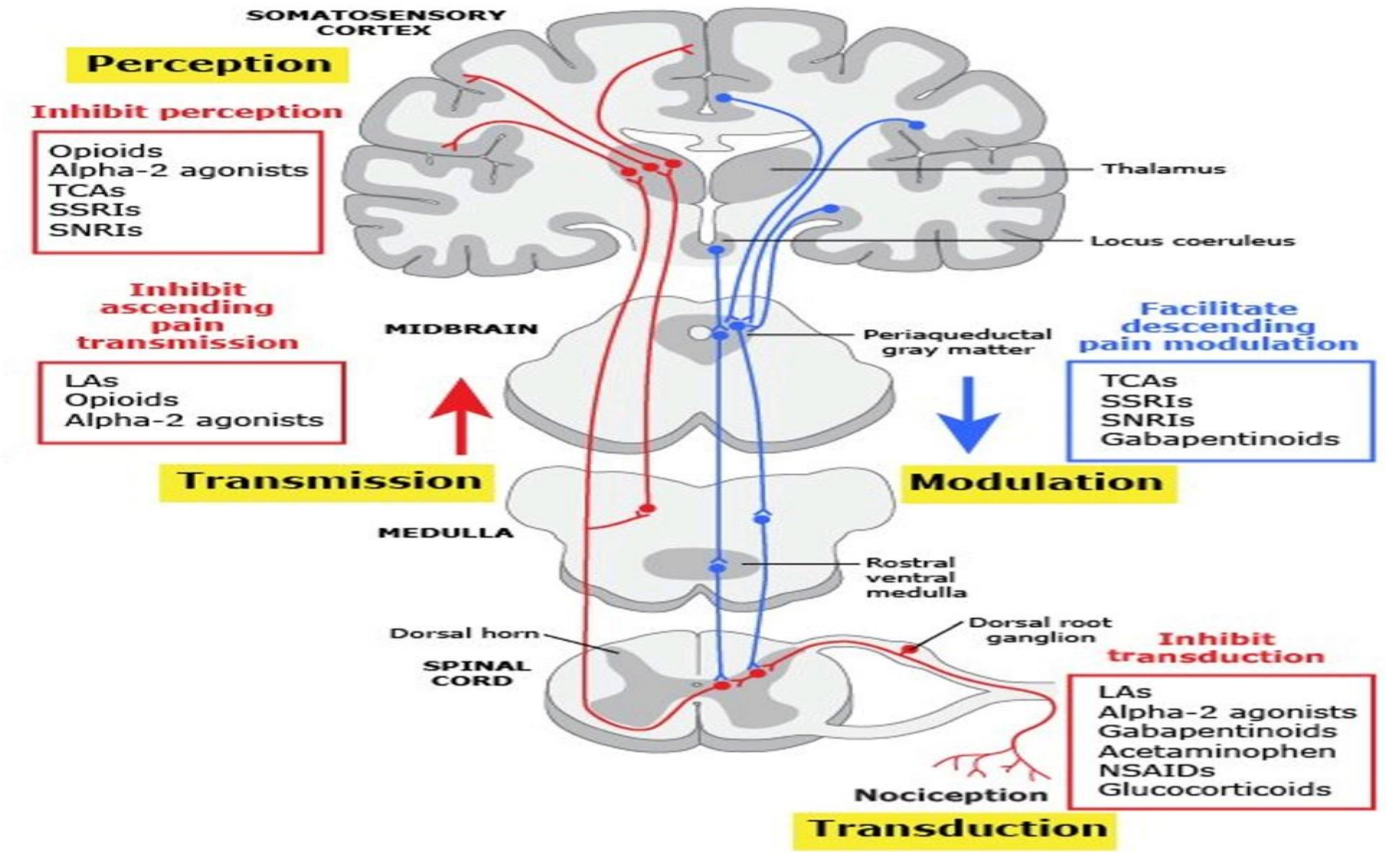


FIGURE 13-4 Pain-transmission and modulatory pathways. ***A***. Transmission system for nociceptive messages. Noxious stimuli activate the sensitive peripheral ending of the primary afferent nociceptor by the process of transduction. The message is then transmitted over the peripheral nerve to the spinal cord, where it synapses with cells of origin of the major ascending pain pathway, the spinothalamic tract. The message is relayed in the thalamus to the anterior cingulate (C), frontal insular (F), and somatosensory cortex (SS). ***B***. Pain-modulation network. Inputs from frontal cortex and hypothalamus activate cells in the midbrain that control spinal pain-transmission cells via cells in the medulla.

Pain pathways and mechanisms



This graphic shows the four major processes of pain transmission and what are thought to be the primary sites of action of medications that affect those processes.

TCA: tricyclic antidepressant; SSRI: selective serotonin reuptake inhibitor; SNRI: serotonin-norepinephrine reuptake inhibitor; LA: local anesthetic; NSAID: nonsteroidal anti-inflammatory drug.

- The most inclusive model of pain is **the "Biopsychosocial Model"** which provides the most exhaustive explanation of pain etiology. This theory of pain hypothesizes that pain is a result of the intricate interactions between biological, psychological, and sociological factors. It was not until 1977 that the biopsychosocial model was scientifically investigated to explain the etiology of some medical conditions. It supports the notion that the human body cannot be compartmentalized when considering management options for pain. John D. Loeser, an anesthesiologist, was the first person to implement this model . Loeser proposes that four parts needed to be taken into consideration in the treatment of pain: nociception (nociceptive component), pain (sensory-discriminatory component), suffering (motivo-affective component), and pain behaviors (the cognitive behavioral component) . The way we approach pain management today is largely based on Loeser's four elements of pain, and failure to consider these elements can be considered an inadequate assessment of care. The biopsychosocial model of pain is the closest to incorporating genetic factors.

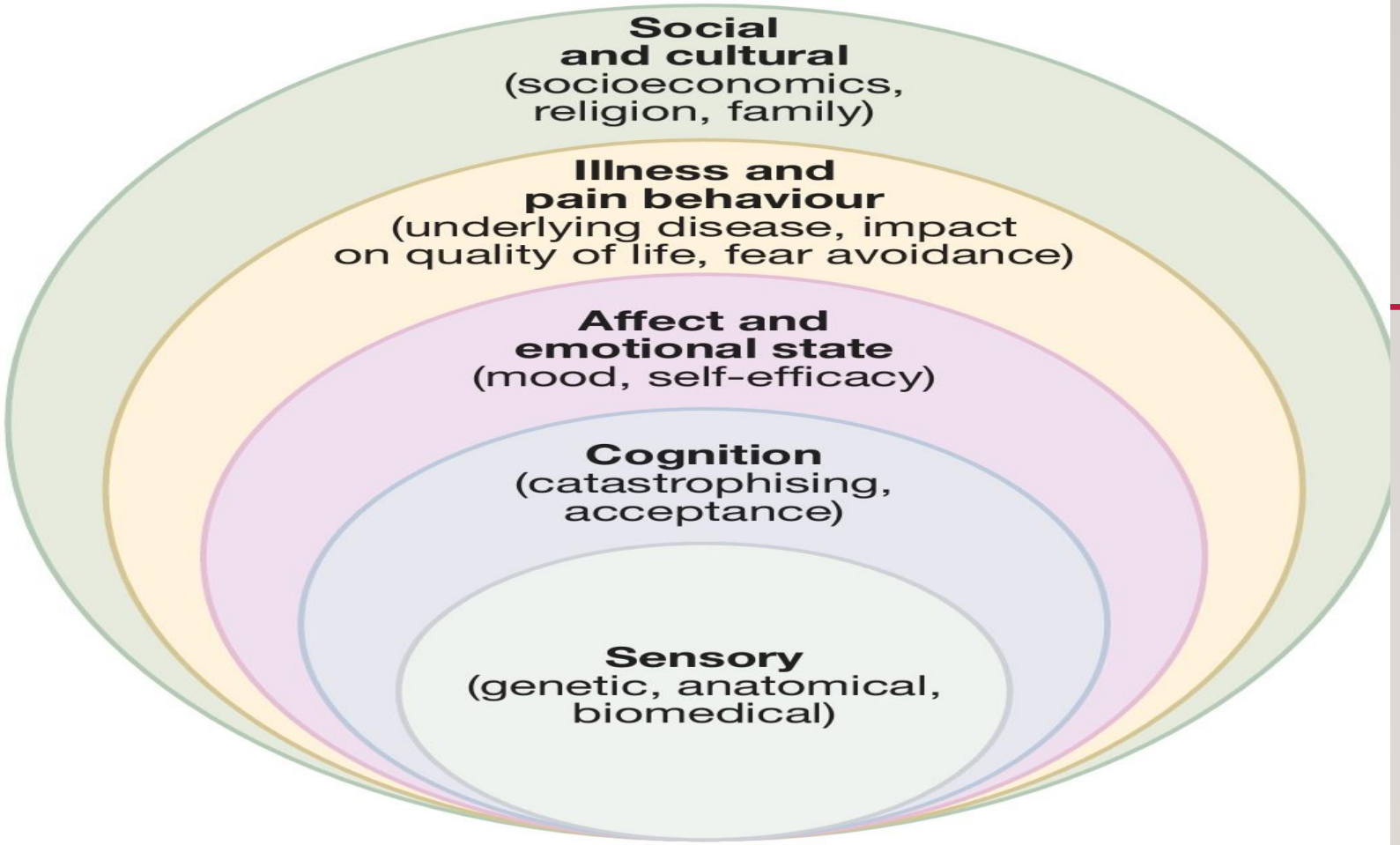
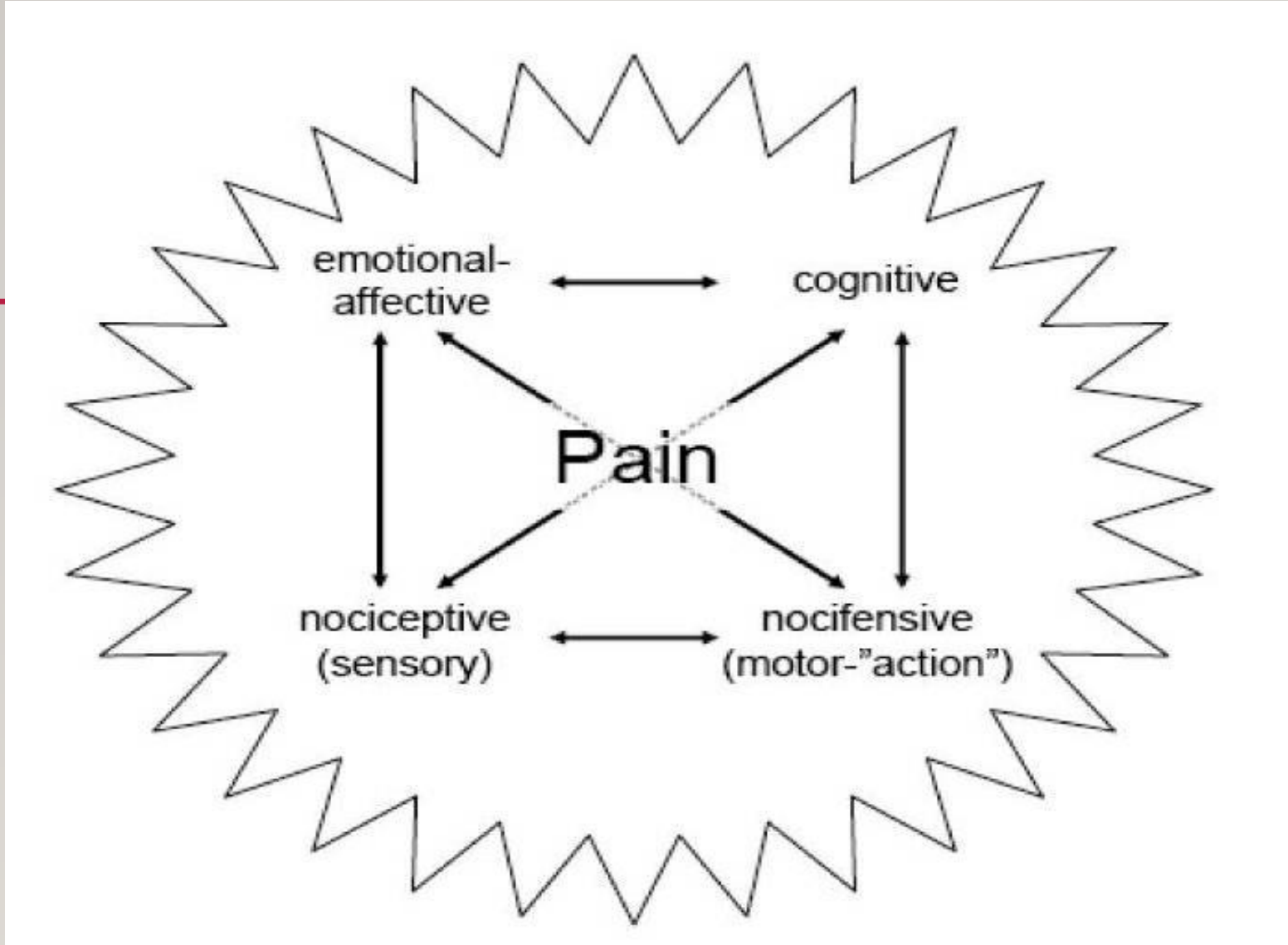


Fig. 34.1 The biopsychosocial model of pain. The perception of pain as a symptom is dependent not only on sensory inputs but also on the individual's cognitive reaction to the pain, their emotional state, their underlying disease and their social and cultural background.


emotional-
affective
cognitive
Pain
nociceptive
(sensory)
nocifensive
(motor-"action")

Intensity Theory
Plato (c. 428 to 347 B.C.) defined pain as an emotional response to intense stimuli.

Cartesian Dualistic Theory
René Descartes (1596-1650) proposed that pain could independently result from psychological factors or physical injury but not synergistically.

Specificity Theory
Charles Bell (1774-1842) introduced the brain as an abstract structure that assigns specific input pathways to each of the different sensations.

Johannes Müller (1801-1858) contributed to the specificity theory by associating individual sensations with their respective receptors.

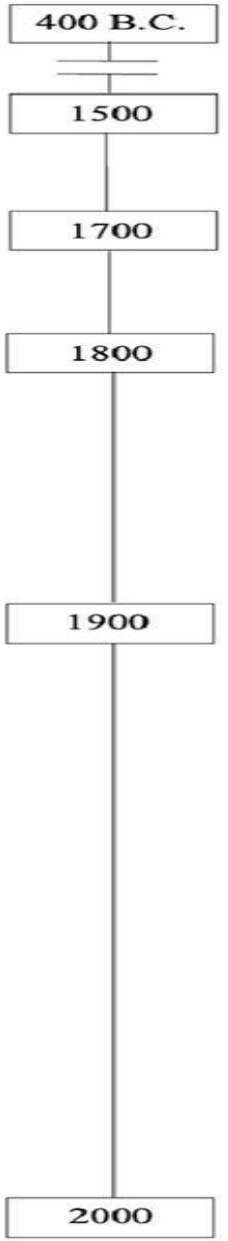
Maximilian von Frey (1852-1932) also advanced the specificity theory by introducing a "pain center" located in the brain that registers the pain signals transmitted from their respective receptors.

Pattern Theory
John Paul Nafe (1888-1970) built on the specificity theory by explaining that there are no individual receptors for the four sensory modalities. Instead, each sensation relays a specific pattern of pain impulses to the brain that is then registered.

Gate Control Theory
Patrick David Wall (1925-2001) and Ronald Melzack (1929-2019) were the first to introduce pain perception as a mind-body experience. This theory states a neurological gate within the dorsal horn opens in response to painful stimuli. If non-painful stimuli overcome the painful stimuli, the gate will close, and the individual will not experience pain.

Neuromatrix Model
Ronald Melzack's experience with phantom limb pain led to the theory that the origin of pain lies within the CNS rather than relayed through to the PNS.

Biopsychosocial Model
John D. Loeser a was the first to implement this model. He hypothesized that pain perception resulted intricate interactions between biological, psychological, and sociological factors.

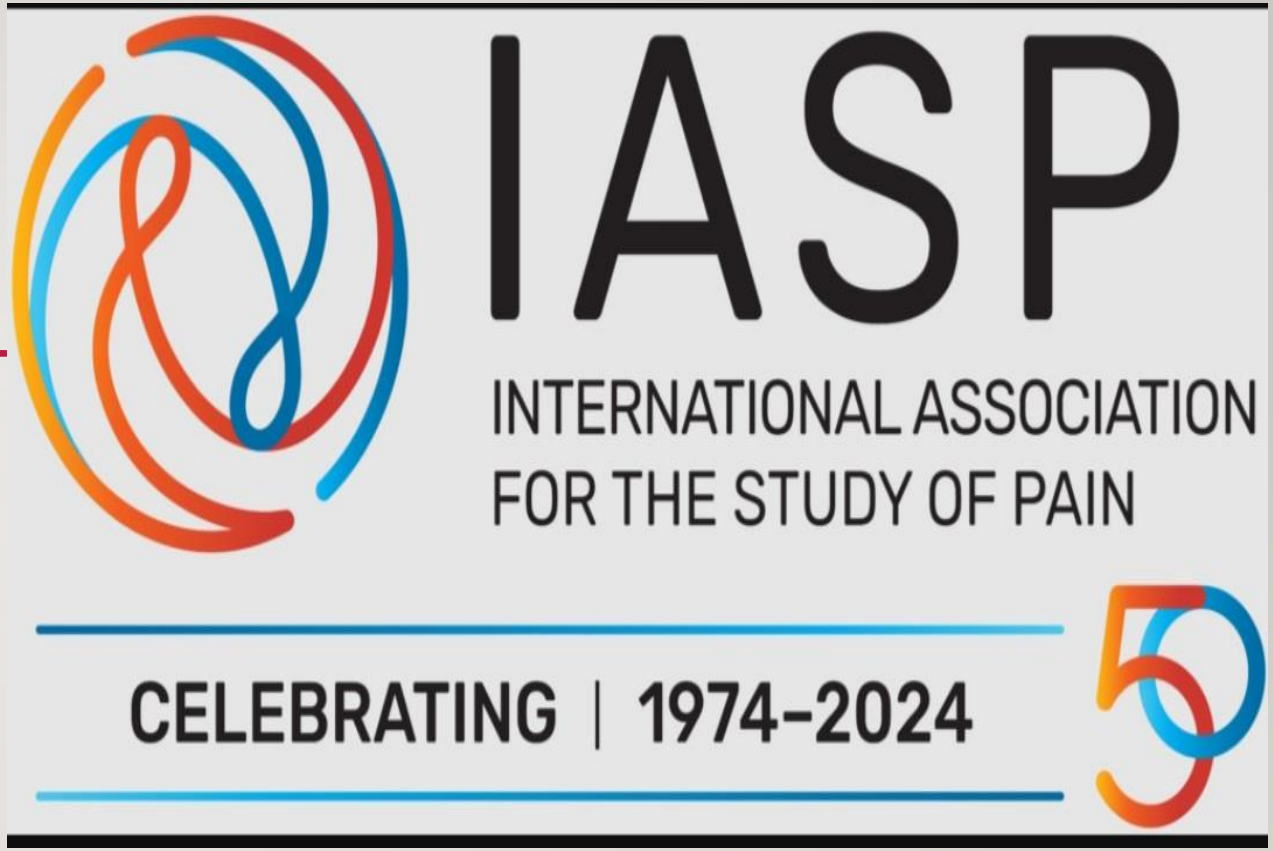


YOUR MIND IS NOW TRYING TO UNDERSTAND ALL INFORMATION SIMILAR TO THIS ?..

What is Pain?

DEFINITION OF PAIN

- **"an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage.**



ASSESSMENT OF PAIN

- ."Pain is a subjective and unique perceptual experience with multiple dimensions that may not be directly observed by others or measured by physiological tests. Pain assessment depends significantly on self-report.
- Objective quantification of pain has been one of the greatest challenges physicians have faced in modern medicine.
- any pain of moderate or higher intensity is accompanied by anxiety and the urge to escape or terminate the feeling. These properties illustrate the duality of pain: it is both sensation and emotion. When it is acute, pain is characteristically associated with behavioral arousal and a stress response consisting of increased blood pressure, heart rate, pupil diameter, and plasma cortisol levels.

The inquest was heard at Winchester Coroner's Court (Alamy Stock Photo)

Man with severe toothache killed himself after 'he had enough of the pain'

Anthony Hoskins had been on the NHS waiting list to get his wisdom tooth removed but his mum was saving up to pay for private treatment after seeing the pain he was in

By **Martyn Halle, Lorraine King**
10:49, 21 Oct 2019

The pain produced by injuries of similar magnitude is remarkably variable in different situations and in different individuals.

Pain must be assessed with a multidimensional approach Especially for chronic pain

2.2 Characteristics of pain (SOCRATES)

Site
- Somatic pain, often well localised (e.g. sprained ankle)
- Visceral pain, more diffuse (e.g. angina pectoris)

Onset
- Speed of onset and any associated circumstances

Character
- Described by adjectives (e.g. sharp/dull, burning/tingling, boring/stabbing, crushing/tugging) preferably using the patient's own description rather than offering suggestions

Radiation
- Disease progressing locally
- Referred by a shared neuronal pathway to a distant unaffected site (e.g. diaphragmatic pain at the shoulder tip via the phrenic nerve [C_3, C_4])

Associated symptoms
- Visual aura accompanying migraine with aura
- Numbness in the leg with back pain suggesting nerve root irritation

Timing (duration, course, pattern)
- Since onset
- Episodic or continuous:
  - If episodic, duration and frequency of attacks
  - If continuous, any changes in severity

Exacerbating and relieving factors
- Circumstances in which pain is provoked or exacerbated (e.g. eating, coughing)
- Specific activities or postures, and any avoidance measures used to prevent or limit pain
- Effects of specific activities or postures, including effects of medication and complementary medical remedies

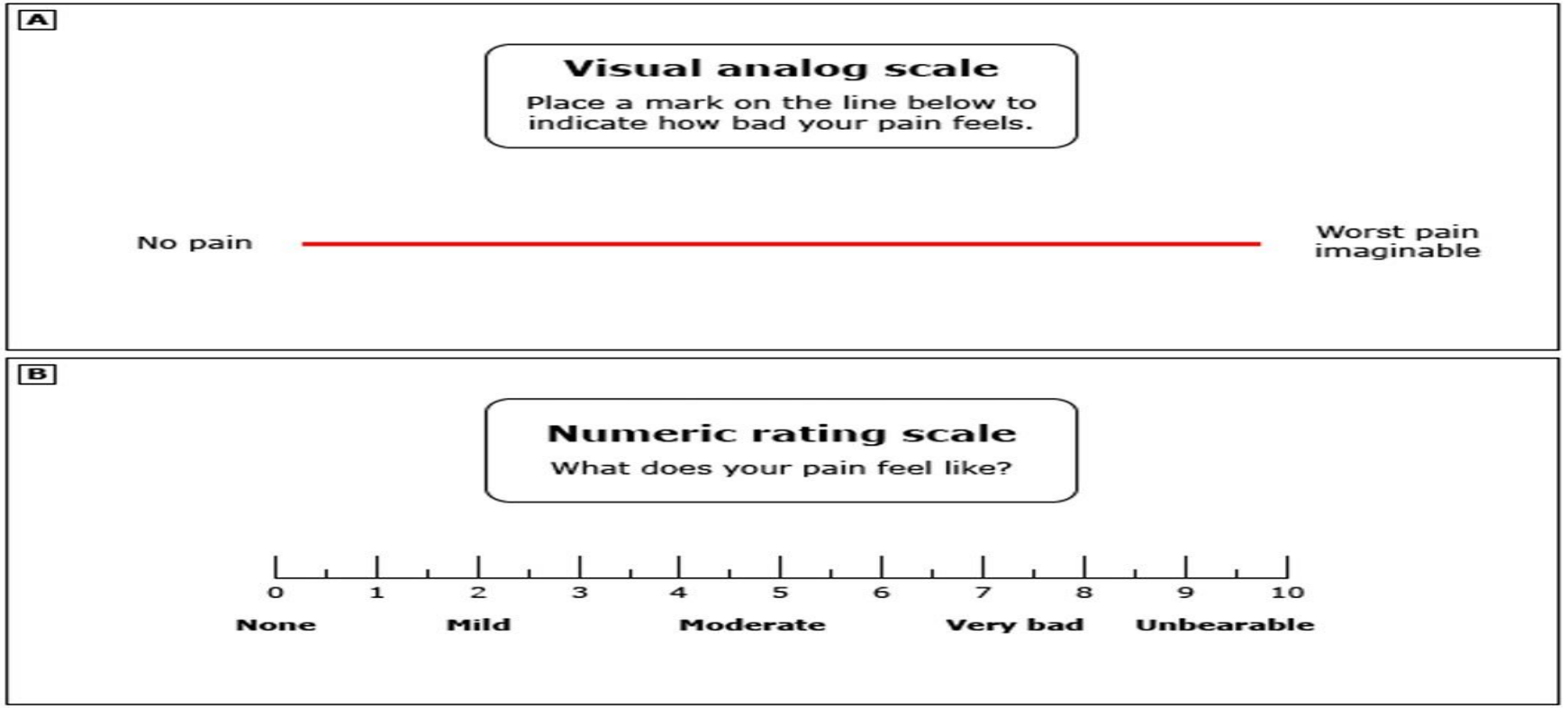
Severity
- Subjective, but ask patient to rate from 1 (just present) to 10 (worst imaginable)
- Sometimes helpful to compare with other common pains (e.g. toothache)
- Variation by day or night, during the week or month (e.g. relating to the menstrual cycle)

#SINGLE-DIMENSIONAL SCALES

- Verbal Rating Scales (Verbal Descriptor Scales) utilize common words (eg, mild, severe) to grade pain intensity. The Melzack and Torgerson scale uses five verbal descriptors: mild, discomforting, distressing, horrible, and excruciating.
- Numeric Rating Scales are common and simple.

Patients may be asked to circle numbers equally spaced on a page or verbally rate pain intensity using a scale of 0–10, in which 0 represents "no pain" and 10 represents "the worst pain imaginable." Advantages of numeric scales are their simplicity, reproducibility, and sensitivity to small changes in pain.

Visual analog and numeric rating pain scales



(A) When using a VAS, the patient is asked to mark a 10 cm line at a point that corresponds to the degree of pain. The VAS score is the distance in millimeters from the left end of the line to the patient's mark.

(B) When using an NRS, the patient indicates the number that corresponds to pain severity, either verbally or by marking the scale.

VAS: visual analog scale; NRS: numeric rating scale.

CHILDREN, ADULTS, PATIENTS WITH MILD TO MODERATE COGNITIVE IMPAIRMENT, AND PATIENTS WITH LANGUAGE ISSUES

Wong-Baker FACES pain rating scale

Explain to the child that each face is for a person who feels happy because they have no pain (hurt) or sad because they have some or a lot of pain. Face 0 is very happy because they don't hurt at all. Face 1 hurts just a little bit. Face 2 hurts a little more. Face 3 hurts even more. Face 4 hurts a whole lot. Face 5 hurts as much as you can imagine, although you don't have to be crying to feel this bad. Ask the child to choose the face that best describes how they are feeling. The rating scale is recommended for persons age 3 years and older. Point to each face using the words to describe the pain intensity. Ask the child to choose the face that best describes their own pain, and record the appropriate number.

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PAIN ASSESSMENT IN INFANTS:

Infants are dependent on their caregivers to assess their pain and to determine the effectiveness of management efforts because they cannot verbalize their pain sensations. Facial activity, crying, and body movements are the most studied behavioral responses to pain in neonates.

Two tools use a combination of behavioral and physiological measurement. **CRIES** (ie, crying, requires oxygen, increased vital signs, expression, sleeplessness) uses the five variables on a 0–2 point scale to assess neonatal postoperative pain. **The Modified Behavioral Pain Scale uses three factors** (facial expression, cry, and movements) and has been validated for 2- to 6-month-old children.

PAIN ASSESSMENT IN THE ELDERLY

- An important factor in pain assessment in the elderly is assessing the effect the pain is having on their lives, rather than the intensity of the pain itself. Necessary activities of daily living are often maintained despite severe pain.

Table 3.3 Functional Activity Scores

Score	Comments
A	No limitation of relevant activity
B	Mild limitation of relevant activity
C	Severe limitation of relevant activity

Source: Adapted from Scott and McDonald (2008).
Note: The score is assessed relative to the patient's baseline functional ability and based on activity that is relevant to the cause of the acute pain.

PATIENTS WITH SEVERE DEMENTIA

Pain Assessment in Advanced Dementia (PAINAD) scale

Items*	0	1	2	Score
Breathing independent of vocalization	Normal	Occasional labored breathing. Short period of hyperventilation.	Noisy labored breathing. Long period of hyperventilation. Cheyne-Stokes respirations.	
Negative vocalization	None	Occasional moan or groan. Low-level speech with a negative or disapproving quality.	Repeated troubled calling out. Loud moaning or groaning. Crying.	
Facial expression	Smiling or inexpressive	Sad. Frightened. Frown.	Facial grimacing.	
Body language	Relaxed	Tense. Distressed pacing. Fidgeting.	Rigid. Fists clenched. Knees pulled up. Pulling or pushing away. Striking out.	
Consolability	No need to console	Distracted or reassured by voice or touch.	Unable to console, distract, or reassure.	
			Total¶:	

This pain assessment score can be used to assess pain in patients with dementia. Patients should be observed for 5 minutes prior to performing the assessment. Total scores range from 0 to 10, with 10 representing severe pain.

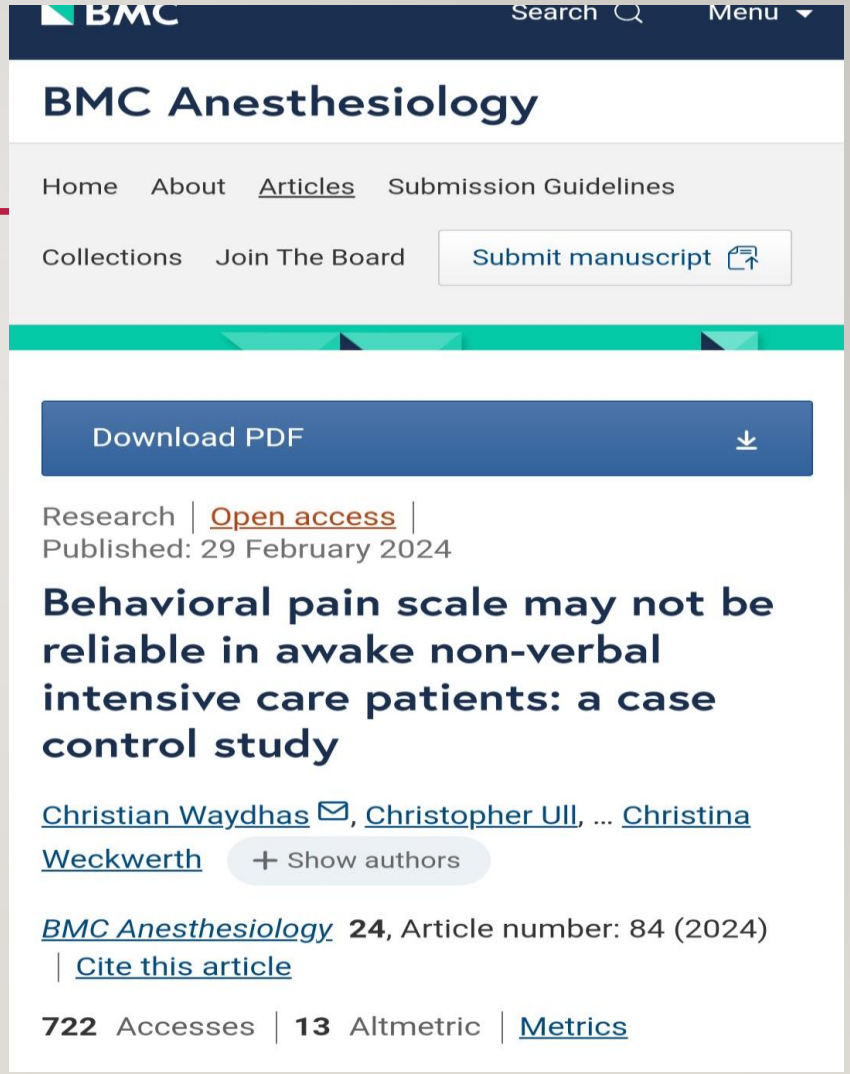
* 5-item observational tool.

¶ Total scores range from 0 to 10 (based on a scale of 0 to 2 for 5 items), with a higher score indicating more severe pain (0 = "no pain" to 10 = "severe pain").

CRITICAL ILL PATIENTS?

Critical care pain observation tool

Indicator	Description	Score	
Facial expression	No muscular tension observed	Relaxed, neutral	0
	Presence of frowning, brow lowering, orbit tightening, and levator contraction	Tense	1
	All of the above facial movements plus eyelid tightly closed	Grimacing	2
Body movements	Does not move at all (does not necessarily mean absence of pain)	Absence of movements	0
	Slow, cautious movements, touching or rubbing the pain site, seeking attention through movements	Protection	1
	Pulling tube, attempting to sit up, moving limbs/thrashing, not following commands, striking at staff, trying to climb out of bed	Restlessness	2
Muscle tension	No resistance to passive movements	Relaxed	0
Evaluation by passive flexion and extension of upper extremities	Resistance to passive movements	Tense, rigid	1
	Strong resistance to passive movements, inability to complete them	Very tense or rigid	2
Compliance with the ventilator (intubated patients)	Alarms not activated, easy ventilation	Tolerating ventilator or movement	0
	Alarms stop spontaneously	Coughing but tolerating	1
	Asynchrony: blocking ventilation, alarms frequently activated	Fighting ventilator	2
OR			
Vocalization (extubated patients)	Talking in normal tone or no sound	Talking in normal tone or no sound	0
	Sighing, moaning	Sighing, moaning	1
	Crying out, sobbing	Crying out, sobbing	2
Total, range			**0-8**



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QUALITY IMPROVEMENT | VOLUME 25, ISSUE 2, P152-159, APRIL 2024

Evidence-Based Pain Assessment in Nonverbal Palliative Care Patients

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Published: January 20, 2024 •

DOI: https://doi.org/10.1016/j.pmn.2023.12.005 •

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PlumX Metrics

TABLE 2. MOPAT DATA IN ALERT AND ORIENTED (A&O) AND COGNITIVELY IMPAIRED/NONRESPONSIVE (CI/NR) PATIENTS

Subscale and items	*A&O patients (n = 28)*[a]	*CI/NR patients (n = 7)*[a]	*CI/NR patients (n = 23)*[b] *Time 1 (before pain intervention)*	*CI/NR patients (n = 22)*[c] *Time 2 (after pain intervention*
Behavioral	*Pain*	*Indicators*		
Restless	None 61% Mild 36% Moderate 3% Severe 0%	None 57% Mild 43% Moderate 0% Severe 0%	None 13% Mild 22% Moderate 35% Severe 30%	None 46% Mild 36% Moderate 14% Severe 4%
Tense muscles	None 36% Mild 53% Moderate 11% Severe 0%	None 29% Mild 57% Moderate 0% Severe 14%	None 0% Mild 13% Moderate 44% Severe 44%	None 23% Mild 59% Moderate 18% Severe 0%
Frowning/Grimacing	None 36% Mild 50% Moderate 14% Severe 0%	None 57% Mild 14% Moderate 14% Severe 14%	None 0% Mild 13% Moderate 70% Severe 17%	None 64% Mild 23% Moderate 9% Severe 4%
Patient sounds	None 90% Mild 10% Moderate 0% Severe 0%	None 43% Mild 43% Moderate 14% Severe 0%	None 4% Mild 17% Moderate 61% Severe 17%	None 27% Mild 64% Moderate 4.5% Severe 4.5%
Mean (SD)	2.07 (0.87)	3.0 (3.3)	8.1 (1.95)	2.1 (2.57)
Physiological	*Pain*	*Indicators*		
Blood pressure	No change 82% Change 18%	No change 71% Change 29%	No change 57% Change 43%	No change 86% Change 14%
Heart rate	No change 75% Change 25%	No change 71% Change 29%	No change 22% Change 78%	No change 73% Change 27%
Respirations	No change 86% Change 14%	No change 86% Change 14%	No change 22%, Change 14%	No change 64% Change 35%
Diaphoresis	No change 96%, Change 4%	No change 100%, Change 0%	No change 48% Change 52%	No change 77% Change 23%
Mean (SD)	0.61 (0.87)	0.71 (0.95)	2.5 (1.47)	1.0 (1.02)

[a]Sample from Project 2.
[b]Sample from Project 3.
[c]$n = 22$ due to missing data.

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- Davidson 24
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